

strated CR. MCE and SPECT showed sensitivities of 80% and 75% ($p=NS$) respectively for the detection of CR, the specificities were also similar (65% vs. 69% respectively). MCE had an improved specificity (84%) when only segments with homogenous contrast opacification were considered viable compared to 78% with normal tracer uptake on SPECT. On a patient basis, the extent and intensity of contrast opacification by MCE was significantly higher in the 25 patients with CR when compared to those without ($p=0.008$), no significant difference was noted in SPECT ($p=0.06$). MCE was superior to SPECT for predicting an improvement in regional ventricular function in the infarct related area ($p=0.002$ vs 0.02). **Conclusions:** MCE was superior to SPECT for the identification of myocardial viability in patients following AMI. MCE is a valid alternative to SPECT for the assessment of viability following AMI.

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Quantification of Absolute Myocardial Perfusion by Contrast Echocardiography: Algorithm, In Vitro and In Vivo Validation

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Background: Absolute tissue perfusion (ml/min/g) is the gold standard for the functional assessment of the microcirculation. To date, absolute myocardial perfusion in humans can only be obtained by positron emission tomography (PET). Based on a volumetric model of ultrasound contrast agent (UCA) kinetics for the description of UCA refill curves following ultrasound-induced microsphere destruction, we developed an algorithm for absolute perfusion measurements using myocardial contrast echocardiography (MCE). Objectives of this study were to validate this algorithm in vitro (1) and in vivo (2) as well as to demonstrate its generalization (3).

Methods: The spatial form of the continuity equation yielded absolute perfusion: $\Pi = \beta \times \mu / \rho$, where ρ is tissue density. Parameter β and μ indicate the rate constant of rise of the refill curve and the intravascular volume ratio (IVR) that were calculated from refill sequences recorded by real-time MCE during constant intravenous UCA infusion. (1) A hemodialysis filter mimicking microcirculation (IVR=0.213) was perfused with saline by a pump at flow rates between 10 and 200 ml/min. (2) Assessment of regional myocardial perfusion in 14 healthy volunteers by MCE and PET. (3) Evaluation of regional renal and cerebral perfusion in a healthy volunteer by MCE.

Results: (1) For flow velocities from 0.4–8.6 mm/s, mean (SD) μ was 0.219 (0.023). Parameter β correlated linearly with pump flow Q_{pump} ($r^2=0.98$). Spatial integration of Π assessed by MCE yielded total phantom volume flow Q_{MCE} that served as an estimate of Q_{pump} ($Q_{\text{MCE}}=0.90Q_{\text{pump}}+0.45$, $r^2=0.96$, $SEE=8.26$). (2) Perfusion analysis by MCE was successful in 182 of 224 segments. Linear regression analysis showed good agreement between MCE and PET perfusion data ($\Pi_{\text{MCE}}=0.902\Pi_{\text{PET}}+0.076$, $r^2=0.88$, $SEE=0.134$). (3) Renal cortical and cerebral perfusion was 2.91 ml/min/g and 0.91 ml/min/g, respectively, and were in good agreement with published data. **Conclusion:** MCE in conjunction with the presented algorithm allows the measurement of absolute myocardial perfusion and, likely, absolute renal and cerebral perfusion and thus may offer new capabilities to study cardio-, reno- and cerebrovascular diseases.

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Long-Term Prediction of Coronary Events According to the Results of High Dose Dipyridamole Contrast Echocardiography Myocardial Perfusion Studies

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BACKGROUND: High dose dipyridamole contrast echocardiography with intermittent harmonic imaging (MCE) has shown high sensitivity and specificity for diagnosis of myocardial perfusion defects (MPD) in comparison with simultaneous sestamibi SPECT. The prognostic value of MCE to predict coronary events was not studied yet. **PURPOSE:** To demonstrate the prognostic value of reversible perfusion defects detected by dipyridamole MCE's, to predict mortality and coronary events. **POPULATION AND METHODS:** 50 pts from 2 laboratories referred to dipyridamole SPECT were studied using MCE and simultaneous sestamibi SPECT and then followed up for 39 months (25–46 months) looking at death, AMI, unstable angina, cardiac surgery or PTCA. Pts cohort had 32 males, medium age 68 y/o \pm 11 y/o. 2 nd Harmonics with Mechanical index 0.5–0.7 and 1/1, 1/3 and 1/5 end systolic triggering was applied, in apical 2, 3 and 4 chambers views, before and after 0.84 mg/kg dipyridamole injection. MCE's perfusion data were analyzed blindly from SPECT results. MPD were considered when defects persist beyond the 1/3 trigger interval. SPECT perfusion defects (SPD) were independently recognized by nuclear cardiologists. Reversible MPD and SPD were those perfusion defects present only after dipyridamole injection. **RESULTS:** 9 pts had events during follow up (2 deaths, 11 PTCA, 3 cardiac surgery and 8 unstable angina). There were 26 pts without any MPD who had 4 events; 15 pts with fixed MPD showed 1 event (NS). 9 pts had reversible MPD and showed 4 events $p < 0.02$ vs patients without MPD and fixed MPD. 24 pts without any SPD had 3 events; 16 pts had fixed SPD and showed 2 event (NS). 10 pts with reversible SPD showed 4 events $p < 0.04$ vs pts without SPD and fixed SPD. Kaplan Meier actuarial survival curves showed differences in events rate since 3 months after the studies, for reversible MPD and SPD (Log Rank test $p < 0.04$ and $p < 0.05$ respectively) and fixed SPD or MPD did not showed significant differences in comparison with pts without any defects. **CONCLUSION:** Reversible perfusion defects detected in high dose Dipyridamole stress MCE, are predictive for coronary events.

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Tissue Inflammation Impairs Tissue-Level Perfusion and Promotes Left Ventricular Remodeling in Patients With Acute Myocardial Infarction

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Background: It has been reported that an increase in C-reactive protein (CRP) level is associated with left ventricular (LV) remodeling in patients with AMI, but its underlying mechanism remains unknown. We hypothesized that inflammation impairs capillary function and, thus, impairs healing process of infarct myocardium, resulting in LV remodeling. **Methods and Results:** We assessed tissue-level perfusion with myocardial contrast echocardiography in consecutive 114 patients with first Q-wave AMI admitted within 24 hours of onset of AMI. They underwent primary coronary intervention. Serial measurements of serum CRP levels were performed every 24 hours. Left ventriculography was performed 4 weeks later to measure left ventricular end-diastolic volume index (LVEDVI). LVEDVI was significantly higher in the no-reflow group than the good-reflow group (66 \pm 18 versus 59 \pm 12 ml/m², $p < 0.01$). The no-reflow group had higher CRP level than the good-reflow group (10.0 \pm 4.6 versus 6.4 \pm 4.7 mg/dl, $p < 0.001$). Re-elevation of ST segment after reperfusion was more frequently found in the no-reflow group than the good-reflow group (63 % versus 26 % $p < 0.001$). Multivariate logistic analysis showed that peak CRP and ST re-elevation were independent determinants of no-reflow phenomenon.

Conclusion: Tissue inflammation along with reperfusion injury impairs tissue-level perfusion. Microvascular dysfunction may play an important role on the development of LV remodeling after reperfused AMI.

Multivariate Logistic Regression analysis for No-reflow phenomenon

	Relative risk	95% CI	P value
PCPK	1.00	1.001-1.002	0.0001
ST re-elevation	8.45	1.45-49.81	0.018
Anterior MI	5.73	1.31-24.86	0.021
Peak CRP	1.34	1.04-1.73	0.024
Hyperlipidemia	0.49	0.11-2.20	0.348
Wall motion score(day-1)	1.03	0.88-1.20	0.750
Killip>2	0.95	0.09-10.26	0.966

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Collateral Blood Flow in the Presence of the Persistently Occluded Infarct-Related Artery Can Be Accurately Predicted by Myocardial Contrast Echocardiography

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Background: Adequate collateral blood flow (CBF) at rest can sustain myocardial viability despite persistent occlusion of the infarct related artery (IRA) in acute myocardial infarction (AMI). This has both therapeutic and prognostic implications. Studies addressing the value of intravenous myocardial contrast echocardiography (MCE) to detect CBF after AMI in humans are limited.

Methods: Seventy consecutive patients with AMI underwent low-power intravenous MCE using Sonovue[®] infusion 7–10 days after thrombolysis. Myocardial perfusion detected by MCE was analysed (qualitatively and quantitatively) in the akinetic segments in 20 (29%) patients with occluded IRA who subsequently underwent revascularization. Contractile reserve, which is a marker of myocardial viability, was assessed with low dose dobutamine 12 weeks after mechanical revascularization.

Results: Of the 102 (32%) akinetic segments, 37 (36%) showed contractile reserve. Contractile reserve was present in 24 (83%) of the 29 segments with homogenous contrast opacification and absent in 60 (82%) of the 73 segments with reduced/absent opacification. Quantitative peak contrast intensity, myocardial blood velocity and the myocardial blood flow were significantly higher ($p < 0.0001$) in the segments with contractile reserve when compared to those without contractile reserve (fig).

Conclusion: MCE may be used as a reliable bedside technique for the accurate evaluation of collateral blood flow in presence of an occluded IRA after AMI.

